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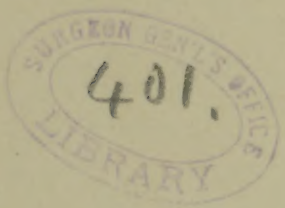

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CAN WE DIAGNOSTICATE HYPERÆMIA OR ANÆMIA OF THE BRAIN AND CORD?*

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Hyperæmia and anæmia of the brain and cord are terms that are very glibly used by the general practitioner to explain many symptoms whose pathology is obscure to him, although the use of the terms by the specialist has steadily decreased within the last ten or fifteen years. Can such hyperæmia or anæmia be diagnosticated? is a very important question that we may well ask ourselves in the interests of precise medicine. It will be instructive in this regard to review the opinions of recent writers of standing.

Gowers speaks with great skepticism of cerebral congestion, or the possibility of the recognition of this condition. The author only speaks of cerebral anæmia as recognizable when it is due to general anæmia, cardiac weakness, intracranial pressure, and obstruction of some cerebral artery. Of anæmia and hyperæmia of the spinal cord he says: "The occurrence of variations in the state of the ves-

* The presidential inaugural address read before the New York Neurological Society, Tuesday, May 6, 1890.

sels of the cord, and the effects that such variations may produce, are matters of inference from symptoms observed during life—symptoms that are in themselves open to various interpretations. Where the ground is barren of facts, theory is always luxuriant." Seeligmüller, in 1887, speaking of cerebral anæmia and hyperæmia, says: "The differential diagnosis between the two, remarkable as it may seem, is often accomplished with great difficulty, although it is of great importance in a therapeutic sense. A chlorotic girl may display the same ruddy cheeks, the same sensation of pulsation in the brain, the same vertigo, the same excitement as a person who is threatened with cerebral hæmorrhage. Even other diagnostic signs fail one, such as the amelioration of the anæmic headache by putting the head in a dependent position, or after a hearty meal. If one examines more precisely, however, in most cases of cerebral anæmia the pallor of the skin and the visible mucous membranes, the blowing murmur of the heart and the large vessels, the small pulse, etc., can readily be distinguished from the lively injection of the conjunctiva and other mucous membranes and the full and heaving pulse of cerebral hyperæmia. If, in addition, there should be found a hypertrophy of the left ventricle, the diagnosis would be so much safer. In general, we can state the signs of differential diagnosis to be as follows:

"Of cerebral hyperæmia: Insomnia, myosis, cephalalgia, extended headache and vertigo, better in standing; hyperæmia of the retinal vessels; pulse slow and full.

"Of cerebral anæmia: Drowsiness, mydriasis, headache restricted to a small space; headache and vertigo better in lying; anæmia of the retinal vessels; pulse small and rapid; heart and venous sounds."

In spinal hyperæmia he would have the symptoms to consist of certain dull pains and slight sensory derangements in

the lower extremities, spontaneous muscular twitchings and tremor, and slight exaggeration of reflexes; and he quotes with approval Hammond's statement that vesical paresis and erections may occur frequently. The main differential diagnosis, in Seeligmüller's opinion, is from the pain of gall-stones. Spinal anæmia is not mentioned by this author. Liebermeister (1886), speaking of cerebral hyperæmia, says: "As symptoms, which perhaps are deducible with a certain probability from simple arterial hyperæmia, may be mentioned these: Cephalalgia of pressing or beating type, excitability and irritability, intellectual and bodily unrest and excitement, with impulse to activity, increased energy of the movements, greater vivacity of the association of ideas and thought, and insomnia. But we must freely admit of these symptoms that they are, to a certain extent, theoretically constructed, and that for the most of them the proof of their dependence upon simple hyperæmia without other disturbance has not been made." Of cerebral anæmia he speaks with more certainty, and the causes which he alleges are obstruction of cerebral arteries, great loss of blood, general anæmia, cardiac disease, and organic intracranial disease. Strümpell (1887), speaking of cerebral hyperæmia and anæmia, says: "In many cases in which pronounced cerebral symptoms point to an abnormal condition of the cerebrum, but where we can, nevertheless, for many reasons, exclude a gross anatomical lesion, we can assume the existence of circulatory disturbances in the brain without being able to adduce a direct reason. So we may attribute to cerebral circulatory disturbances certain cases of headache, pressure in the head, vertigo, general hyperæsthesia, and that Protean and yet well-characterized symptom-group which is known as cerebral neurasthenia. But how far cerebral circulatory disturbances here play a part, what their nature is, and whether purely functional disturbances of the brain may

not arise entirely independent of them, has not yet been positively determined." Writing of the spinal circulatory disturbances, he says: "Our knowledge of the occurrence and the clinical significance of pure circulatory disturbance in the spinal cord is very scanty. All that is stated in this regard in the descriptions of spinal pathology largely corresponds more to theoretic assumptions than to real objective facts." Ross (1882) admits that "the symptoms of cerebral anæmia are not unlike those of hyperæmia of the brain, and the delirium from anæmia which arises in the course of acute disease may very readily be mistaken for the delirium of active congestion. Anæmia and hyperæmia of the brain can indeed only be distinguished from one another by careful attention to the concomitant symptoms. The symptoms themselves are not to be relied upon, as the delirium in anæmia may be as violent as in congestion, and the color of the face is not always a faithful index of the condition of the cerebral circulation. The diagnosis must be founded upon the general history of the case, the nature of the concomitant symptoms, and the treatment which has been adopted prior to the onset of the delirium. As further aids to the diagnosis, it may be tried whether the erect or horizontal posture has any influence in aggravating or diminishing the symptoms, and whether they are increased or relieved by alcoholic stimulants. The state of the general circulation must also be carefully examined." All that he says in regard to the diagnosis of spinal anæmia is this: "The diagnosis must rest mainly on the concomitant symptoms, the symptoms of the acute ischæmic form resembling those due to spinal hæmorrhage, and anæmia can only be inferred to be the cause when the aorta is known to be obstructed or a great loss of blood has recently occurred. The chronic forms of spinal anæmia resemble chronic myelitis or chronic meningitis, but when severe general anæmia

exists it may be inferred that the disease is caused by it. The fact that the horizontal position relieves the symptoms may afford valuable aid in forming the diagnosis," which latter point he borrows from Dr. Hammond. Of hyperæmia of the cord he says: "The diagnosis of hyperæmia of the structures within the spinal canal is based on the slight and transitory nature of the sensory and motor symptoms, the absence of increase of temperature, the short and favorable course of the symptoms, and the success of treatment calculated to relieve congestion." Of all contemporary authors, however, Dr. William A. Hammond is the most precise, enthusiastic, and comprehensive in his account of the symptoms of cerebral anæmia and hyperæmia. Writing (1886) of cerebral congestion, he treats of the active and the passive forms separately. Of the first he recognizes six varieties, which are designated by the chief feature characterizing the attack—namely, the apoplectic, the paralytic, the convulsive, the soporific, the maniacal, and the aphasic; "the latter," he says, "being a sixth form which is now for the first time systematically arranged in the present category." He goes on to say: "It will doubtless be the case that, as our knowledge of the functions of the brain becomes greater, other forms of cerebral congestion, especially those of a partial character, like the aphasic, for instance, will be recognized." In cerebral anæmia the symptoms given by Dr. Hammond are syncope; headache limited to a small spot or consisting of a sense of constriction; tinnitus aurium; dilated and sluggish pupils; small retinal vessels; pale chorioid; paresis of ocular muscles; great facial pallor; nausea and vomiting in extreme cases; epileptiform convulsions occasionally, and always when there has been great loss of blood; general muscular weakness or general or partial paralysis; anæsthesia; mental disturbances, varying from complete coma through the

gradations from low delirium to great mental irritability or intellectual lassitude; hallucinations and illusions; drowsiness; rapid death; a melancholic tendency, or even positive insanity. The symptoms of spinal congestion, according to Dr. Hammond, are: a dull, aching pain, increased by the recumbent posture and by standing, if the lower cord is affected, but not augmented by pressure; an occasional sensation of heat in the cord; disturbances of sensibility and motility, such as hyperæsthesia, shooting pains, girdle sensation, formication, etc.; erections, vesical and rectal paralysis, diminution of electro-muscular reaction, and bedsores. Of spinal anæmia Dr. Hammond writes: "A deficient quantity of blood in the spinal cord, or a depreciation of the quality of the blood circulating through its tissue, give rise to cognate but, so far as their phenomena go, different affections. In one of these, which has heretofore been known as spinal irritation, the morbid action is in a great measure confined to the posterior columns of the cord; in the other, which embraces several differently named disorders, characterized by paralysis—such as reflex paralysis, inhibitory paralysis, spinal paresis, paralysis from peripheral irritation, etc.—the antero-lateral columns are mainly affected. In thus specifically locating the lesions in these affections, I am aware of the fact that post-mortem examinations are wanting to support them. Nevertheless, the symptoms characteristic of each are so distinctly marked, and are in such intimate physiological relation with the regions of the cord specified, that I do not think I am at all exceeding the limits of probability."

All the authors quoted are writers of the last decade, and their views are fairly representative of the views held by neurologists in general, except that those of Dr. Hammond would, I think, be regarded as extreme. Among the older writers the expressions in favor of hyperæmia and

anæmia of the brain and cord are much more marked, but it would profit us little to spend any time in learning what their opinions were, because they classed under these general names so many diseases that have since been differentiated.

It may be said, then, that the symptoms alleged to be those of cerebral hyperæmia or anæmia are headache, flushing or pallor of the face, delirium, vertigo, motor or sensory paralysis, retinal changes, slight aural changes, insomnia, myosis. If these symptoms are examined one by one, the significance of each one will become decidedly problematical to any one who has had much acquaintance with the clinical aspects of cerebral disease. Headache, for example, is a very general symptom, indicating too many disorders to be of any service except in conjunction with other signs, and the statement that it is apt to be localized and widespread in cerebral hyperæmia is a pure assumption, of which no proof has ever been adduced. The condition of the capillary circulation of the face is by no means a reliable guide to the condition of the cerebral capillary circulation. Any hospital surgeon knows that marked meningeal hyperæmia is often associated with great facial pallor in fractures of the skull and the attendant cerebral hyperæmia, just as great pallor may exist with many hyperæmic intracranial conditions, at the same time that precisely the same pathological states may cause vascular flushing in other individuals. Delirium, like headache, is a general symptom indicative of too many conditions to be of any value by itself. The same remark is true of vertigo. Of motor and sensory paralysis as symptoms of cerebral hyperæmia and anæmia it may simply be said that there is not an authentic case on record in which a simple anæmia or hyperæmia has caused a motor or sensory paralysis. It is almost, if not absolutely, impossible to distinguish retinal changes consisting

only of slight differences in vascularity from the normal, as every ophthalmologist knows, while the more marked changes of neuro-retinitis have never yet been shown to have been produced by cerebral anæmia or hyperæmia. Tinnitus aurium and insomnia, like headache, delirium, and vertigo, are symptoms that are absolutely worthless by themselves. Myosis is said to be a symptom of cerebral hyperæmia. It may be, but nobody has yet made public the proof. It will thus be seen that, of all these symptoms, headache, delirium, vertigo, tinnitus aurium, and insomnia are general symptoms which only derive a diagnostic value from their associates, while flushing or pallor of the face are extremely doubtful indications of the intracranial circulation; motor and sensory paralysis and retinal changes are absolutely worthless as signs, and the diagnostic value of myosis is a pure assumption. Anæmia and hyperæmia of the cord have, as we have seen, very lack-luster defenders among the modern writers, with the exception of Dr. Hammond. The former ascribe to them certain vague pains, which might be due to so many varying general, muscular, and neuralgic conditions that the differential diagnosis would be practically impossible, even if it were of any practical use. But when we come to consider the symptoms which Dr. Hammond alleges to be those of cerebral hyperæmia, we are confronted by the picture of an organic cerebral disease. If cerebral hyperæmia can produce the symptoms of apoplexy, paralysis, convulsions, a soporific condition, mania, and aphasia, it certainly is capable of producing serious organic symptoms, and we should naturally expect to have the proof. When we turn to Dr. Hammond's chapter on the pathology of this particular form of cerebral disease we find the statement that the capillaries and large blood-vessels of the brain and pia mater will be found to be increased in size, that the white matter of the brain is increased in consistence

and density, the gray matter is red or even violet in hue, there is a large amount of subarachnoid effusion or even an effusion into the ventricles, and the chorioid plexuses are often enlarged. If there have been repeated attacks of cerebral congestion, granules of hæmatin will be found in contact with the blood-vessels, or the latter may be found to be unusually tortuous and to have minute aneurysms. Now, I submit that this description is not that of a mere hyperæmia, but is a condition of actual structural disease of the cerebral tissue quite as pronounced as can be found in many cases of general paresis of the insane, or in the severer cases of chorea, or in certain cases of intracranial syphilis; indeed, it would seem that a part of this description—that relating to the hæmatin and the tortuosity and aneurysmal swellings of the arterioles—is acknowledged to be from the work of Laborde upon Softening and Congestion of the Brain of the Aged, and written in 1866. In addition to this description, Dr. Hammond also says that we will find that state of the brain described by Calmeil in 1826, and subsequently given the name of *l'état criblé* by Durand-Fardel in 1854, and described respectively in treatises upon the insane and the aged. Dr. Hammond's symptoms of spinal congestion are those of myelitis, and there is no means of making the differential diagnosis, while that spinal anæmia may be confined to the posterior columns of the cord, giving rise to the symptoms of spinal irritation, or confined to the anterolateral columns of the cord, giving rise to paralytic symptoms, is a theory which he himself acknowledges to be wanting in the support of autopsies.

The truth of the matter is that our knowledge of nervous diseases has increased so rapidly within the last quarter of a century as to make it a matter of little surprise that the older authors should have attempted to satisfy their ignorant consciences by dubbing as congestion or hyperæ-

mia the many puzzling symptoms which time has resolved into distinct symptom groups. The most difficult of all phrases for the average scientist is, "I do not know." A disease must be labeled at any cost, and, once labeled, it takes many years to rub out the brand. Meningitis, general paresis, porencephalitis, cerebral and spinal syphilis, disseminated sclerosis, bulbar lesions, poliomyelitis anterior, central or transverse myelitis, syringomyelitis, progressive muscular atrophy, acute ascending or Landry's paralysis, locomotor ataxia, especially in its early stages, many forms of neuritis, focal diseases of the brain in the light of our modern knowledge of localization, lateral sclerosis, many forms of lithæmia and neurasthenia—these are many diseases of which we now possess a knowledge at which the practitioner of twenty-five years ago might well stand appalled, and which he may well be pardoned for supposing to be due to some vague form of anæmia or hyperæmia. Indeed, Leyden, writing in 1875 of essential infantile paralysis, thought it to be due to a peculiar congestion of the cord, while Cornil and Lépine had demonstrated some two years before that the pathological condition was a disappearance of the great ganglion cells in the anterior horns of the spinal cord, the autopsy in their case being followed by another one to the same effect by Gombault in the same year as Leyden's article.

After this necessarily cursory review we may ask ourselves again, Can hyperæmia or anæmia of the brain and cord be diagnosticated? I, for my part, would answer very positively that the diagnosis is not possible by means of the nervous symptoms alone; in other words, the symptoms as I have narrated them above are not sufficient by themselves to warrant a diagnosis of anæmia or hyperæmia of the brain or cord. It must be remembered that the brain is an exquisitely and almost inconceivably complex organ,

pulsating and vibrating with the most explosively nitrogenized molecular life, and reacting to a myriad reflex influences in the kaleidoscopic way that constitutes the never-tiring charms of character, poetry, music, and the varied forms of mental activity. To put our finger on a few slight changes of temporary nature that occur in an organ like this, say that they are really due to hyperæmia or anæmia, and exclude all the cellular play, all the reflex influences from the other viscera of the body, all the morbific elements that may be circulating in the blood, all the factors of temporary discomfort that are to be found in the atmosphere, in the food and clothing, and in the circumstances of a human being's environment—even if this is not impossible, it has certainly not been done. If we have evidences of some intracranial disturbance, such as those we have already detailed—headache, delirium, vertigo, tinnitus aurium, insomnia, flushing or pallor of the face—and with these we have some concomitant conditions that would make it reasonable to suppose that there might exist a congestion or anæmia of the brain or cord, then we should be warranted in regarding such a diagnosis as probable. Thus certain cardiac conditions, lesions of the great vessels of the chest, abdomen, and thorax, certain forms of hepatic disease, tumors making pressure upon these great vessels, a general anæmia, leucocythæmia, intense mental exertion, or exposure to great heat—these conditions, when associated with the above-described general symptoms, would make it probable that the general symptoms were due to hyperæmia or anæmia, as the case might be; but even then there might not be certainty. I can not conceive, however, how we are to make the diagnosis of spinal anæmia or hyperæmia, for I know of no pathological observations that would warrant on the one hand the diagnosis by means of the vague pains that are described by some authors, or, on the other hand,

by means of the symptoms of full-fledged myelitis which are described by Dr. Hammond.

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